with tobacco amblyopia and nutritional (tropical) amblyopia at risk in that hydroxocobalamin, and not cyanocobalamin, is a powerful cyanide antagonist. If such patients are given cyanocobalamin, the diagnosis may be questioned and the patient be thus condemned to a life of poor sight.

Patients with tobacco amblyopia who have normal serum vitamin B12 levels need not continue therapy with systemic hydroxocobalamin once their visual acuity and visual fields have returned to normal providing they abstain from further smoking. However, patients who have low serum vitamin B12 levels and evidence of defective vitamin B12 absorption will need to continue indefinitely with hydroxocobalamin as will all patients with proven pernicious anaemia who are at risk of developing optic neuropathy if they are, or later become, smokers.

I would remind Dr Lederle that besides being present in tobacco-smoke and alcohol, cyanide has a worldwide distribution in the plant kingdom as well as being present in industrial waste. There may thus come a time when more widespread chronic cyanide neurotoxicity occurs from a dietary source in persons with a genetic or acquired error of cyanide or vitamin B12 metabolism and that such patients will only respond to treatment with systemic hydroxocobalamin[5].

A G FREEMAN
Meadow Rise
3 Lakeside
Swindon SN3 1QE

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I should like to correct an error in the paper by Dr Freeman (November 1992 *JRSM*, pp 686-7). Since at least the fifth *World Health Organization Model List of Essential Drugs*, published in 1988, hydroxycobalamine, not cyanocobalamine, has been the drug included.

MARCUS M REIDENBERG
The New York Hospital
Cornell Medical Center
525 East 68th Street,
Box 70, New York, NY 10021, USA

The author replies below:

I do not dispute the data supplied by Professor Reidenberg but this, with respect, was not the point at issue.

In 1970 we presented a case for withdrawal of cyanacobalamin in favour of hydroxocobalamin for therapeutic use. As no action was taken by the manufacturers, we again asked in 1978 'why has cyanacobalamin not been withdrawn?'. This view received widespread support. Yet, until 1988, the World Health Organization's Committee on the selection of essential drugs continued to list cyanacobalamin to the exclusion of hydroxocobalamin, thus placing an incalculable number of patients with tobacco and nutritional amblyopia and optic neuropathy in pernicious anaemia at risk.

A G FREEMAN
Meadow Rise
3 Lakeside
Swindon N3 1QE

He/she

I refer to the correspondence in the *Journal* (by Ellis and Ridgway (April 1992 *JRSM*, p 246), Ellerby and Lindsey (September 1992 *JRSM*, p 586) and Jackson (October 1992 *JRSM*, p 651) regarding the necessity for a sex-neutral personal pronoun in English. The solutions proposed by Ellerby (s/he) and Ellis (hshe) could be used in writing, but their spoken pronunciation is not obvious. Lindsey's suggestions are syntactically ingenious, but unpronounceable, and at odds with the historical tendency of English usage to eliminate case differences.

I therefore propose the neologism 'esh' as the case-insensitive, sex-neutral personal pronoun so clearly needed in late twentieth century English, and look forward to its enthusiastic and trend-setting adoption by *JRSM* in place of the inflammatory sex-specific pronouns and of the awkward circumlocutions often used to avoid them.

SIDNEY N KAHN
1425 Grasshopper Road
Huntingdon Valley, PA 19006, USA

Nitrous oxide at homeopathic doses

I would like to comment on some of the issues raised by Dr R Schoental in her recent letter (September 1992 *JRSM*, p 587). A number of studies on the cognitive consequences of trace concentrations of nitrous oxide (in the region of 500 ppm), have revealed an absence of effects, although mood was not specifically examined[1]. None the less, analgesic concentrations of nitrous oxide can be antidepressant in certain subjects[2]. The effects of the homeopathic concentrations alluded to by Dr Schoental, have, as far as I am aware not been examined for their mood altering effects. However, there is no evidence that concentrations of nitrous oxide as low as in the ppb range can have any effect on folate and cobalamin metabolism[3,4].

There is another point; nitrous oxide as an anaesthetic is not especially used for paediatric dentistry. Today, as previously, it is employed, as an anaesthetic for all forms of surgery, both general and dental. None the less, it is also used in dentistry, obstetrics and for treating addictive withdrawal states at analgesic doses in which there is no loss of consciousness[2,5].

M A GILLMAN
South African Brain
Research Institute
6 Campbell Street
Waveley 2090 Johannesburg
South Africa

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5 Gillman MA, Lichtigfeld FJ. Analgesic nitrous oxide for alcohol withdrawal: a critical appraisal after 10 years' use. *Postgrad Med J* 1990;**66**:543-6

The author replies below:

I am delighted to learn that Dr Gillman is interested in the effects of pharmacological concentrations of nitrous oxide in neuropsychiatry. He may be able to establish the relation of the concentrations of this compound in the atmosphere to the factors that determine human mood, a problem which I am not able to undertake, but appears to warrant appropriate studies.

It may be worth noting that in a study of the global and seasonal variability of the composition of stratosphere and of the mesosphere by the experimental meteorological satellite Nimbus 7 (launched in 1978), only the data for nitrous oxide (N_2O) appeared to differ from those expected from a prediction model[1].

R SCHOENTAL
15 Birdhurst Court,
Woodcote Road,
Wallington,
Surrey SM6 0PG

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Post anoxic action myoclonus

We read with interest the recent report of Rustom and Fisken (December 1992 *JRSM*, p 761) on post anoxic action myoclonus. Increased awareness of this condition, on both the intensive care unit[1] and general medical wards, is to be welcomed as a prerequisite for appropriate treatment, but we would take issue with some of the authors' statements regarding its pathophysiology and treatment.

Despite low levels of CSF metabolites of 5HT, not all patients with this syndrome respond to serotoninergic drugs[2]. Furthermore, although, as stated, clonazepam is the drug of choice, the evidence available is against its action being at 5HT receptors[3], indeed its effects (reduced 5HT utilization in brain) are exactly the opposite of those predicted from the beneficial effects of 5-hydroxytryptophan in human myoclonic disorders[3]. Hence, an action at GABA receptor complexes seems more likely[3], although why clonazepam should be more efficacious than other benzodiazepines is unclear. CSF GABA levels are low in post anoxic myoclonus, and GABA agonists such as baclofen have occasionally been reported of benefit[4].

Contrary to the implications of Rustom and Fisken, clonazepam does not always produce a dramatic therapeutic response; other agents (a wide variety of which have been tried[5]) may need to be added, especially sodium valproate[2]. Another drug which may be useful is piracetam (2-oxo-1-pyrrolidine acetamide): it is claimed to be of value in some patients with cognitive deficits, especially of hypoxic origin, and on the basis of its hypothesized effects on energy metabolism has been tried in post anoxic myoclonus. Its chemical structure is similar to GABA but its precise mechanism of action is unknown[6]. We recently treated a 56-year-old man who suffered anoxic brain damage following a respiratory arrest during a severe asthma attack; his myoclonic movements were not well controlled by clonazepam (10 mg/day) and sodium valproate (4 mg/day), but addition of piracetam (UCB SA Pharmaceuticals, Brussels, Belgium; 7.2 g/day) produced considerable amelioration with marked improvement in the performance of the activities of daily living. Other reports of the efficacy of piracetam have appeared[2,7-9], and it seems of particular use in patients with evidence of cortical (as opposed to reticular) reflex myoclonus.

A combination of drugs may therefore be necessary in severe post anoxic myoclonus[2]. Since the site, severity, and extension of hypoxia-induced damage is variable between patients, differing responsiveness to drugs is not surprising and suggests the need for an empirical approach to therapy.

ANDREW J LARNER
M TOM E HEAFIELD
University Department
of Clinical Neurology
The University of Birmingham,
Edgbaston, Birmingham B15 2TH

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